Effect of Dust on the Lungs*

JUDGING from the number of investigations carried out during the past few years on the incidence and physiological effects of dust on persons employed in various industries, consideration of the hazard is assuming more and more importance in the minds of those concerned with public health.

The present report deals principally with the incidence of silicosis and does not take into account other health hazards that may be present in the dusty trades.

OCCURRENCE OF SILICOSIS

This disease, which is distributed in all parts of the world, is found in many industries, such as metal mining, the quarrying and dressing of granite, sandstone, millstone, and flint, the refractory industry, and in sandblasting.

In an investigation into the health and working conditions of employees in the mining industry of Australia carried out in 1928,[1] it was found that silicosis and tuberculosis, either singly or combined, exist as industrial diseases among the employees of the mining industry in Tasmania, as well as in Victoria. Gold mining in Victorian mines is a much more potent cause of industrial pulmonary disease than work in the Tasmanian copper, silver-lead, and tin mines.

Recently in a report on "The Present Position of Silicosis in Britain," *The Lancet*[2] quoted statements by Dr. Middleton, the medical inspector of factories, calling attention to the occurrence of the disease in various industries throughout the country.

According to Dr. Middleton the sandstone industry represents the most widespread of all silicosis producing industries, but the incidence of the disease is diminishing owing to the application of water and mechanical methods of dust suppression. The granite industry, although allied to sandstone quarrying and dressing, is complicated because of the varying proportions of quartz, feldspar, and mica present in the rock. As the result of an inquiry into the slate industry there appears to be some evidence of the production of fibrosis of the lungs, but the occurrence of actual disability within the ordinary working lifetime of workers is not clearly shown.

In the grinding of metals, silicosis, and especially the combined disease of silicosis with tuberculosis, was at one time extremely prevalent, but the sandstone wheel is being largely replaced by wheels of abrasives such as carborundum. Allied to the grinding of metals is the process of sand blasting metals and etching

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glass, by playing a jet of sand under high pressure against a metal or glass surface to produce certain effects. The process is becoming more extensively used, and it is a matter for serious consideration whether a substitute such as iron or steel grit could not be used to replace the siliceous grit as the abrading material. In pottery manufacture, both in the manufacture of earthenware and of china, Dr. Middleton suggested that " potter's asthma " is due to dust containing ground flint. Ground silica is used in many industries, and, in the crushing and grinding of the silica and in the industries in which ground silica is used, silicosis, sometimes of an acute type, occurs. The evolution of dust in these industries is controlled by the use of mechanical methods of handling, enclosure of machinery, and the use of localized exhaust draught.

Tin mining in Cornwall has had a high mortality rate from tuberculosis, and it has been shown that this is associated with silicosis. Drilling of the highly siliceous rock in which the tin ore occurs is now done through hollow steel with a water feed. Silicosis also occurs in coal miners employed on hard headings.

The report also states that the ganister and silica bricks manufactured in the refractories industry contain over 90 per cent of silica and that a medical board composed of specialists periodically examines each of the 2,000 or 3,000 persons employed in this industry.

According to Stewart[3] in districts of Lancashire County, England, where the industry of coal mining predominates (40 per cent of the occupied males being engaged in mining), the male death rate from pulmonary tuberculosis is higher than the corresponding rate for the whole administrative county. Since the death rate from pulmonary tuberculosis among all coal miners of England and Wales is below that for the general population, the statistical evidence suggests strongly that there is in Lancashire an occupational factor at work that raises the death rate among miners, and that this factor is silicosis.

An inquiry was made by Sutherland and Bryson[4] to obtain evidence regarding the occurrence of silicosis among workers in England manipulating sandstone in connection with the granting of compensation to sandstone workers since February 1, 1929, under the Workmen's Compensation Act of 1925. The findings disclosed that 1 out of every 4 men at work appeared to be the subject of " mason's disease." Since the men were all examined at work, any case so far advanced as third stage, and also many in the second stage, would not be found at work.

In an investigation of health conditions in a sandstone paving stone quarry, which had been in operation near Paris for 50 years, it was found[5] by a study of mortality records that for the period 1881 to 1926 all the deaths occurring among the workers were attributed to respiratory diseases.

In Germany, according to Teleky,[6] in spite of outstanding improvement in conditions, the mortality from tuberculosis is very high among metal polishers—higher than among similar mining groups. He considers sandstone to be the most dangerous grinding stone and recommends the substitution of artificial stone and the introduction of other protective measures such as dust removal, reduction of working time, separate rooms for polishing, and medical supervision.

At the suggestion of the county industrial physician, the Tuberculosis Hospital at Schwetzingen[7] examined 23 of the men engaged in sandblasting in a metal polishing plant after it had been observed that several of the workers were affected by lung diseases. Of the sandblasters examined all except 2 complained of troubles. This report emphasizes the finding that the pneumonoconiosis caused by sand dust

is more dangerous than anthracosis. The time of activity of these men in this industry was comparatively short; in most of the cases there was a pronounced roentgenological finding and in some changes of great severity and extent were observed.

An X-ray and clinical examination of 282 workers, who had been employed for 15 years or more in the most dust producing operations of a porcelain factory in the vicinity of Moscow, Russia,[8] showed an unexpectedly low percentage of pneumonoconiosis. This was the more surprising as porcelain dust contains large amounts of SiO_2. Of the 282 workers examined, 24.1 per cent showed suspicious indications of pneumonoconiosis and 13.5 were definitely affected by the disease.

At the meeting of the Permanent International Commission for the Study of Industrial Diseases, which was held in Lyon, France, April 3–6, 1929,[9] Dr. Irvine in his paper, " Diagnosis of Silicosis as an Occupational Disease," gave the following figures regarding the incidence of silicosis among European miners on the Witwatersrand:

> During the past 11 years, 4,092 cases of " simple silicosis," 377 " tuberculosis with silicosis," and 365 cases of " simple tuberculosis " have been detected by the Medical Bureau among the working miners on the Witwatersrand. Reduced to average figures these numbers represent in the case of " simple silicosis " alone an annual " production " of 372 cases among a population of 13,436 working miners; so that the average annual rate of incidence for the whole period was 2.76 per cent.

So far, reported studies of silicosis have been made for the most part on industrial groups in which the existence of silicosis as a hazard was deducible *a priori*. In the Industrial Department of the Vanderbilt Clinic, the occasional worker who appeared, giving a history of rock drilling in subway or tunnel construction work in New York City only, and who showed on X-ray examination of his lungs well-marked evidence of silicosis, aroused speculation as to the extent to which silicosis develops under conditions of excavation and tunnel construction in this city.

Of 208 men engaged in occupations exposing them to rockdust in Manhattan, who were selected for complete study, 57 per cent showed the presence of silicosis: 23 per cent showed radiographic evidence of ante-primary silicosis; 19 per cent of first stage silicosis; 7 per cent of second stage silicosis; and 8 per cent of third stage silicosis.[10]

In a study made of the abrasive industry, Clark[11] found that of a total of 137 workers exposed to large doses of dust, mostly from abrasives, over periods varying from 10 to 42 years, 42 showed no silicosis, 12 showed slight silicotic change, 77 the picture of first stage, and 6 the picture of second stage silicosis. Of the 6, 2 had been exposed to large doses of clay dust containing 9 per cent of free silica, 4 had been exposed to abrasive dust only. There were 11 cases in which the X-ray showed an old inactive tuberculosis process in the lung.

AGE INCIDENCE AND INFLUENCE OF LENGTH OF EMPLOYMENT IN THE DUSTY TRADES

Inquiry into the age incidence of industrial pumonary disease among miners examined in Australia[1] shows that, while pulmonary tuberculosis may occur in early life, silicosis is a disease of slow evolution and does not appear frequently under the age of 40, and a period of at least 10 years in the industry is necessary to produce it. However, reports on other industries, such as sandblasting and the

pottery industry, would indicate that under some conditions the disease appears earlier. In the pottery industry in England[12] it has been found that in occupations where the occurrence of silicosis is high the disease becomes evident at an early stage in the occupational history of the worker. Sutherland and Bryson state[4] that where the risk is present in the sandstone industry, the disease is much more common after 40 years of age and after 20 years of work in the industry.

According to Mavrogordato,[13] on the Witwatersrand, among miners who develop silicosis, the mean duration of exposure before the disease is clinically recognized is from 10 to 11 years, whereas in the British pottery industry it appears to be about 25 years. He states, however, that although dust is easier to control in a pottery than in a mine, the potters start work much younger than do the Witwatersrand miners and continue in the occupation much longer. Nevertheless the actual annual silicosis rate of the potters appears to be about the same as that of the miners.

In the sandblasting industry, Müller[7] found more severe pneumocotic lung changes with proportionately shorter activity not only among older workers but also among the younger ones, although X-ray examination revealed more severe changes in the older people than in the younger when the time of activity in the industry was the same. Thus, the average age of the workers, examined by Müller, who had more severe lung changes, was 45 years, while the average age of the other workers was 32.6. The time of activity in the industry was with all comparatively short—with one worker 8 years, with one 6 years, and with all the rest under 3 years.

In the porcelain industry, according to Kaestle,[14] workers hardly ever show characteristic pneumonoconiosis under 10 years of work; but in the case of older workers with relatively short working time he found the changes to be clearer and more stubborn than in younger employees of similar working time. He considers that the vitality of the whole man and of the respiratory system plays an essential rôle in the prevention of damage and the self-purification of the intruded dust. He also recommends that only those who have reached a certain bodily maturity be employed in this occupation, since the self-purifying ability of the lungs is greater if the worker is not very young or very old.

In the investigation in New York City[10] it was found that 76 men, or 36.5 per cent, had been exposed less than 10 years, while 132, or 63.5 per cent, had been exposed 10 years or more. The shortest exposure in the entire group was 6 weeks; in the ante-primary group it was 3 years; in the first stage group it was 1 year, and in the second and third stage groups it was 9 years. The longest exposure in the entire group was 46 years. In the ante-primary group it was 40 years; in the first stage group, 46 years, and in the second and third stage groups, 44 years.

According to Hamilton,[15] in the enamel-ware industry, in spite of the improvements that have taken place, it is a significant fact that the working life of an enameler is still short, although his wages are good and his hours often less than the average. One large plant points with pride to a veteran who has worked at the trade for 22 years; another has one with 18 years of service. Since the men start in at 18 or 20 years of age, it is plain that the great majority must give it up before they reach the age of 40 years.

From June, 1927, to December 31, 1928, there were made 15,351 physical examinations of men and 400 of women and children at the U. S. Bureau of Mines Health Clinic, Picher, Okla. This year's work so far indicates that there has been

a reduction in the number of silicotics working in the mines in this district. Much of this reduction is due to the silicotics leaving the mines. A great many of these men have either been given suitable treatment in hospitals, sanatoriums, etc., or have been provided with suitable work outside of the mines.

An investigation, begun in 1926, and reported recently,[16] was made by Hayhurst and his coworkers, in one of the largest and deepest sandstone districts in the world, located in Ohio and worked for more than 50 years. The workmen are employed by two quarry companies which market grindstones, scythestones, curbing, flagging, breakwater and building stone, and also furnace sand. The report summarizes the results as follows:

> A stereoscopic X-ray examination was made of each of the 919 workmen, of which number the films of 913 workmen were readable. Of this latter number, pulmonary pathology was found in 55.1 per cent, including 38.5 with silicosis in various stages. The total tuberculosis, however, was but 1.9 per cent. Likewise general disability was low. These findings check relatively closely with those of the tuberculosis statistics for the community. The period for developing silicosis, namely, 16.24 years, is over twice that reported elsewhere. The rock has an average content of from 92.15 to 97 per cent crystalline silica (SiO_2).
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> A study of silicosis reports throughout the world indicates that tuberculosis is usually present in from 20 to 30 per cent or more of the cases of silicosis, but of our 260 men with silicosis only 13, or 5 per cent, had tuberculosis.

The authors suggest that other silicosis studies at this quarry should include a careful analysis of the rock itself, from both chemical and petrographic points of view.

A few years ago, the U. S. Public Health Service[17] began a series of intensive studies on the health of workers in dusty trades. The first study was reported in 1928, and dealt with the health of workers in a cement plant. The report on the second study,[18] dealing with the health of workers exposed to silica dust in the granite-cutting industry, has recently been published.

This report brings out clearly the extent of the hazard under such conditions as existed in the plants studied. Of particular importance is the fact that it was possible, by differentiating occupations on the basis of the amount of dust exposure, to determine within broad limits how much dust of the composition studied can be tolerated by workers without serious deleterious effects. The conclusion was reached that a maximum of dust exposure falling somewhere between 10 and 20 million particles per cu. ft. of air is a desirable limit for dust containing about 35 per cent free silica in the form of quartz. It was also concluded, on the basis of a study made in other plants having local exhaust ventilation systems, that this limit could be reached by the use of economically practicable ventilating devices of this character. The recommendation was made that occupational processes in which little dust is produced be segregated in separate rooms or buildings.

It should be pointed out that the limit established was not found to prevent the occurrence of silicosis. It was found, however, that there seemed to be no particular liability to pulmonary tuberculosis where the concentration of dust was within this limit.

The study was of such a character as to present a rather definite picture of what happens to men working for many years under a dust hazard of the extent described. The salient points may be summarized as follows:

1. The long period of service before the liability to tuberculosis becomes manifest (generally 20 years or more)
2. The sharp correlation between length of exposure to the dust and the prevalence of tuberculosis and also the death rate from this disease
3. The close relation between the extent of dust exposure and the health of the men
4. The universal occurrence of silicosis among the workers
5. The large proportion of workers finally succumbing to tuberculosis
6. The almost invariably fatal form of the disease within a short time after the onset
7. The different character of silicosis as manifested by X-rays compared with that shown where there is exposure to a dust with a much higher content of free silica
8. The location of the tuberculosis lesion, usually basal, where the disease complicates silicosis
9. The absence of deaths from silicosis *per se,* tuberculosis apparently always intervening
10. The failure of workers to recover from their condition upon going into non-dusty trades
11. The high incidence of sickness of a severe nature from causes other than tuberculosis
12. The rising sickness and mortality rates from tuberculosis due to longer use of the hand-pneumatic tool
13. The high death rates at the present time from tuberculosis, compared with normal industrial experience

ETIOLOGY OF SILICOSIS

Miners who have silicosis, according to Tattersall,[2] will be found without exception to have been engaged principally in drilling or hewing stone. The abnormal appearances in the lungs of the gold miner, the stone mason, the quarryman, and the coal miner are similar although the constituents of the dust may vary, indicating that the silica and that alone gives rise to the pathological condition.

According to a paper by Heffernan[2] on the biophysics of silica and the etiology of silicosis, the disease is the result of the local action of hydrated silica upon the pulmonary tissue; this action is physico-chemical in nature and its development depends upon the rapidity with which fresh silica hydrosol is brought into contact with pulmonary tissues; substances, such as alkalis, which favor the formation of silica hydrosol from silica, when added to silica dust accelerate the development of the silicosis; and substances, such as carbon, coal dust or clays, which retard or prevent the formation of hydrosol from silica or which coagulate the hydrosol when formed, retard or prevent silicosis.

In Australia some evidence was noted that silicosis complicated by tuberculosis may occur earlier than uncomplicated silicosis.

Stewart[3] calls attention to the fact that the precise manner in which the silica acts upon the lung tissue and renders that soil favorable for the development of tuberculosis is still a matter of controversy, one view being that the silica acts as a soluble protoplasmic poison, the other view being that colloidal silica is formed which acts in an adsorbent way on the protoplasm of the cells containing the silica, forming a pabulum which is a favorable soil for the growth of tubercle bacilli.

Stewart considers that there is no doubt that when silicosis and tuberculosis are both present, tubercle bacilli are often extremely difficult to obtain; that such a case may proceed to a fatal termination without tubercle bacilli being found, and yet extensive tuberculosis be found post-mortem in addition to the silicosis. According to him, the question of the infectivity of these combined cases of tuberculosis and silicosis is an interesting one. There is a good deal of evidence available pointing to low infectivity as compared with cases of simple pulmonary tuberculosis. The evidence in his own dispensary area (southeast Lancashire) tends to confirm this view, as the number of positive contacts from such cases has been few compared with contacts from patients with simple pulmonary tuberculosis.

Stewart also called attention to the point of controversy as to the priority of the two conditions, whether the silicosis is antecedent to the tuberculosis or *vice versa*. Dr. Watkins-Pitchford in South Africa thinks that the great majority of cases of simple silicosis would not have developed into such but for the presence of a tuberculous element in the lungs. According to Stewart, clinical evidence neither proves nor disproves this view.

In France a histochemical study was made of lungs taken from subjects having lived in the city and never having worked in coal mines[19] and in another study by the same investigators[20] a cytological and histochemical examination was made of the lungs showing 4 typical cases of anthracosis in miners in Courriere. According to these investigators these studies have enabled them to contribute some new results in regard to the much discussed problem of the nature of anthracosis pigment, whether the ferruginous pigment found is derived from blood or from masses of exogenous particles of coal introduced by respiration.

From these studies the following conclusions were drawn: A small part of the pigment encountered in the anthracosis of miners is certainly formed by a substance derived from hemoglobin, yielding red ashes of iron oxide. The other part, by far the more important, is formed by a pigment which disappears by incineration without leaving any residue of iron oxide. This pigment, which is extremely abundant, can be considered either as a non-ferruginous endogenous pigment or as from coal. The authors think that the latter interpretation is correct for the two following reasons: The particles of this pigment are extremely irregular and non-spherical, as those of an endogenous pigment. They coexist always with other particles of a mineral nature, which on incineration leave an abundant residue of calcarious and silicious ashes. This constant existence of silicious minerals and black pigment indicates that this latter is coal. The coal dust is fixed in the lungs exactly as the silicious dust.

According to Mavrogordato, pigmentation facilitates the tracking of dust by the naked eye, but is not an essential part of the changes produced by dust of free silica. If silica particles be injected intravenously, some pigmentation is to be observed on intracellular particles and this pigment is probably a hemoglobin derivative. He considers the point to be rather of interest than importance, as far as silica itself is concerned, and calls attention to the fact that Virchow, until Zenker convinced him to the contrary, regarded pigmentation in dust-phthisis as due to a breakdown of hemoglobin, and not to the dust inhaled. When one turns to the metallic dusts—cobalt, iron or vermilion, for instance—one is reminded of the work of Mallory and others on hemochromatosis and feels that, in these cases, the part played by the breakdown of hemoglobin may be more significant. The presence of carbon in the air does not appear to effect the changes produced by silica other than by coloring them, and the tissue changes, detailed above, according to

Mavrogordato, can be produced by silica alone, and a silicotic lung need show very little pigmentation.

Mavrogordato summarizes[13] the current views on the etiology of silicosis under two headings: 1. Cases of uncomplicated silicosis, in which no infective element is present; and 2. Cases of silicosis complicated by infective processes, of which the most common is tuberculosis. He asserts that certain industries are " phthisis producing " and defines such a dust under four headings as follows: (a) Its nature, (b) size of particles, (c) faculty of being arrested in the tissues, and (d) concentration of dust, duration of exposure and conditions of exposure.

According to him, a phthisis producing dust arrested in the lungs in sufficient quantity will set up a fibrosis, but will not produce a true " miners' phthisis." The effective occupation of the lungs by a phthisis producing dust facilitates infection by the tubercle bacillus, and may influence the result of infection by certain other organisms. Clinical silicosis and fibrosis in the presence of a phthisis producing dust implies arrest of dust in the lungs. The lungs may, however, be effectively occupied by dust clinically undemonstrable yet capable of influencing an after-coming infection. The disabling and fatal form of miners' phthisis, as now met with on the Witwatersrand, he considers is due to an infection by the tubercle bacillus superimposed upon lungs effectively occupied by a phthisis-producing dust. This event may or may not be preceded by a definite clinical silicosis. Delayed resolution and chronic progressive changes may occur in lungs effectively occupied by a phthisis producing dust after recovery from acute pneumococcal or streptococcal infections.

PHYSIOLOGICAL ASPECTS

There is not much new material along the lines of symptoms, diagnosis, treatment and prevention, efforts of investigators apparently having been devoted mostly to the determination of the presence of the disease in various industries.

Irvine[9] stressed the importance of the infective factor in silicosis and emphasized the fact that it is uncommon to find a condition of clinically obvious tuberculosis present at the stage at which silicosis first becomes detectable. The very great majority of cases of silicosis found among working miners, when first detected, are from the clinical standpoint cases of uncomplicated or " simple " silicosis, without evidence of obvious or " overt " tuberculosis. Nevertheless, " simple " silicosis should not be thought of merely as a dust fibrosis, but as being at least in a majority of cases a dust fibrosis which from its beginning as a clinically detectable condition is linked up with an element of latent tuberculous infection. The after-history of these cases is in this respect significant. In the majority of instances silicosis is a progressive disease. The outlook of the individual case is mainly dependent upon whether the infective element remains " bottled up " and inactive, or whether, on the other hand, it becomes active, or a further infection occurs from outside the lung. In some instances advance is rapid; in others, again, the condition may remain stationary practically indefinitely. But, although there are wide individual differences, dependent no doubt in part upon constitutional, nutritional and environmental factors, yet the tendency of the majority of cases to advance is, on the average, singularly uniform, and the culmination of that advance, if it proceed to its terminal stage, is in a form of obvious active tuberculosis in the silicotic lung, although by no means all cases advance to that condition.

There is some disagreement in regard to the suggested treatment by the administration of some non-silicious dust, such as coal dust, that is apparently harmless and will assist in removing the harmful dust from the lungs.

Dr. Middleton states that it would appear that once enough silica has been inhaled to produce the nucleus of fibrosis, the resulting inhalation of coal dust into a lung in which the lymphatic drainage has been disorganized increases the fibrosis and produces disability.

Kaestle considers that even with the conviction that dust and bacilli can be forced into the respiratory organs in various similar ways, the attempt of such inhalation therapy is driving out the devil with Beelzebub, a very problematical healing of tuberculosis purchased by the certainty of dust lungs.

Mavrogordato states that, according to experimental studies, a phthisis producing dust and an infective agent simultaneously present facilitate each other's action in the direction of producing fibrous tissue. No experimental evidence could be secured suggesting that the presence of coal dust in the lungs exercised a protective action against tuberculosis. The simultaneous inhalation of coal dust and silica dust facilitates the exit of silica laden cells from the lungs, but a lung previously invaded by coal dust has no special ability to rid itself of silica. As already mentioned, a lung invaded by silica retains the fine soot from acetylene lamps.

Müller[7] points out the injustice of not recognizing as occupational the diseases of sandblasters, resulting from their employment, as lead poisoning is in the case of lead workers. However, in England this is done under a scheme of the Workmen's Compensation Act of 1925. In February, 1929, a new scheme became effective known as the Sandstone Industry (Silicosis) Scheme.[4] Some of the provisions of the scheme are that a register, supplied by the employer, must be kept by the worker and produced when required by the employer or the Medical Board, on which shall be entered date of commencement of his employment with employer, particulars of any previous employment in the sandstone industry or in any of the occupations mentioned in the First Schedule to this Scheme, and date of medical examination entered by the Medical Officer making the examination. The workman will be liable to forfeit his right to compensation if he fails to furnish true information or carry out other specified duties under this scheme. In case of a workman employed in the industry on or after April 1, 1929, or in case of death, his dependants will be entitled to compensation, if his death or disability is caused by silicosis. The disease must be due to employment in the industry, and will be so considered if the workman has been employed for a period or periods amounting to not less than 5 years, unless the employer or the company proves the contrary.

If the employer fails to carry out any of his duties he will be liable to pay an increased subscription to the Compensation Fund. The Scheme requires the employers to furnish facilities for the periodic medical examination made at the works and not, without the consent in writing of the Medical Board, to engage or to continue to employ in the sandstone industry any workman who has been suspended under the Scheme or who has refused to submit himself to any examination required under the Scheme. If the workman refuses or wilfully neglects to submit himself to any such examination or in any way obstructs the same, or if after having been suspended he without consent in writing of the Medical Board reëngages in the sandstone industry, he will be liable to forfeit his right to compensation.

All investigators agree that efficient methods of removing or allaying dust, ex-

amination of men before beginning work in the dusty trades and periodically thereafter with the elimination of the tuberculous, are of value in the prevention of silicosis. Although the rule holds that once silica invaded always silica occupied, the lungs have considerable powers of ridding themselves of silica laden cells. This is borne out by observations made on South African native miners[18] who, for the most part, work short contracts interspersed with visits to the kraals and it is surprising how comparatively little naked-eye pigmentation there may be in the lungs of a man who had worked several underground contracts and has afforded opportunities for observation owing to his death from an accident immediately on resuming work after an interval.

According to Mavrogordato, this indicates that intermittent, as opposed to continuous, employment is perhaps the most effective single measure against silicosis. He states that the simple silicosis rate among native miners employed on the Witwatersrand is lower than that among white miners, although the native, when at work, is more exposed to dust than is the white miner. His employment, however, is intermittent as compared with the continuous employment of the white man. A study of a small group of natives who had been employed continuously showed about the same simple-silicosis rate as obtained among white miners; in fact, the mean duration of exposure before disease was recognized in the continuously employed native was less than that in the case of the white man.

SUMMARY

Consideration of the dust hazard in industry, if judged by the reports appearing during the last year, is assuming more and more importance from the standpoint of public health.

The sandstone industry, according to reports, represents the most widespread of all silicosis producing industries. Investigation of this industry in England in connection with the Workmen's Compensation Act showed that 1 of every 4 men at work appeared to be the subject of "mason's disease." Silicosis is found among the metal polishers, metal miners, sandblasters, potters, and any other workers exposed to dust containing free silica. There is a difference of opinion as to free silica being the only causative factor as in New South Wales silicosis has also been found to be caused by orthoclase basalt, which does not contain free silica.

Silicosis may be classed under two headings: uncomplicated silicosis, in which no infective element is present; and silicosis complicated by infective processes, of which the most common is tuberculosis.

Histochemical and cytological studies made on the lungs of miners showing anthracosis and on those of non-miners indicate that the ferruginous pigment found in the lungs of both miners and non-miners is derived from the blood and not from exogenous substances inhaled by them.

Studies made by the U. S. Public Health Service in the granite industry show the close relation between the extent of dust exposure

and the health of the workers. The occurrence of silicosis is universal among them and a large proportion finally succumbs to tuberculosis, death apparently not taking place from silicosis *per se* without the intervention of tuberculosis. The death rate from tuberculosis is high in this industry when compared with normal industrial experience.

South African investigators think that "simple silicosis" should be considered in a majority of cases as a dust fibrosis linked up, from its beginning as a clinically detectable condition, with an element of latent tuberculosis.

A number of authorities stress the progressive character of silicosis since, although there are wide individual differences dependent no doubt in part upon constitutional, nutritional, and environmental factors, the tendency of the majority of cases to advance is singularly uniform and the culmination, if it proceeds to its terminal stage, is in the form of obvious active tuberculosis in the silicotic lung.

There is a difference of opinion in regard to the suggested treatment by the administration of a non-silicious dust, such as coal dust. Middleton thinks that once enough silica has been inhaled to produce the nucleus of fibrosis, the resulting inhalation of coal dust into a lung in which the lymphatic drainage has been disorganized increases the fibrosis and produces disability. Mavrogordato has been unable to secure experimental evidence indicating that the presence of coal dust in the lungs exercises a protective action against tuberculosis but thinks that the simultaneous inhalation of coal dust and silica facilitates the exit of silica laden cells from the lungs, although a lung previously invaded by coal dust has no especial ability to rid itself of silica.

All investigators agree that efficient methods of removing or allaying dust, examination of men before beginning work in the dusty trades and periodically thereafter with the elimination of the tuberculous, are of value in the prevention of silicosis. Although the rule holds that once silica invaded always silica occupied, the lungs have considerable powers of ridding themselves of silica laden cells. According to Mavrogordato this is borne out by observations made on South African native miners who, for the most part, work short contracts interspersed with visits to the kraals and he was surprised how comparatively little naked-eye pigmentation might be in the lungs of a man who had worked several underground contracts and had afforded opportunities for observation owing to his death from an accident immediately on resuming work after an interval.

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